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TITLE: CORNEAL ENDOTHELIAL TOXICITY OF LIPOSOME ENCAPSULATED 5-FLUOROURIDINE IN RABBITS

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Purpose: To investigate the clinical and histological toxicity of the exposure of the rabbit corneal endothelium to anterior chamber injection of liposome-encapsulated 5-fluorouridine (L5-FUR) at concentrations of 1 mg/ml and 0.38 mg/ml and intravitreal injection of L5-FUR at 1 mg/ml.
Methods: A total of 12 New Zealand white rabbits were divided in three groups: A) Anterior chamber injection of 0.1 ml of L5-FUR (1 mg/ml), B) Anterior chamber injection of 0.1 ml of L5-FUR (0.38 mg/ml), C) Intravitreal injection of L5-FUR (1 mg/ml). We treated 3 eyes and 1 control eye (injection of 0.1 ml of empty liposomes) for each group. We studied the clinical corneal edema 24 h after and sacrified the rabbits to study the endothelium using light microscopy (silver stain).
Results: Treated groups: A) We did not observe clinical corneal edema, but we demonstrated a moderate irregular staining of endothelial membranes, a moderate sign of toxicity. B) We did not observe clinical edema, but we demonstrated a lower irregular staining of endothelial membranes, a moderate sign of toxicity. C) We did not observe clinical edema, and endothelial membranes were normal such as the control group.
Conclusions: Anterior chamber injection of liposome encapsulated 5-fluorouridine (L5-FUR) at concentrations of 1 mg/ml and 0.38 mg/ml causes cyclitic membranes and a moderate histological endothelium toxicity whereas intravitreal injection of L-5FUR (0.1 mg/ml) appears nontoxic for the corneal endothelium.

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1H NMR AND IR SPECTROSCOPY OF AQUEOUS HUMOUR OF EYES WITH EMULSIFIED SILICONE OIL

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Purpose: To identify possible detergents in aqueous humour, which may cause silicone oil emulsification *in vivo*, by means of 1H and IR spectroscopy.
Methods: The aqueous humour was aspirated in the beginning of the silicone oil removal from 35 eyes. 1D, 2D NMR and IR spectra of the samples were measured.
Results: Albumin, glucose and lactic acid were the best detectable compounds in obtained samples. 1H NMR spectroscopy failed to find the other proteins in aqueous humour.
Conclusions: 1H NMR and IR spectroscopy is a useful method, which can be used for the study of silicone oil emulsification *in vivo*.
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COMPARATIVE EXPERIMENTAL STUDY OF 10% AND 15% MEDICAL GELATINE SOLUTION IN THE ANTERIOR CHAMBER AND IN THE POSTERIOR EYE SEGMENT AS A VISCOSUBSTANCE IN THE VISCOSURGERY

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Purpose: To assess the application of 15% vs. 10% medical gelatine solution in the anterior chamber and posterior eye segment as a viscosubstance in viscosurgery.
Material and Methods: The experiments were performed on 24 rabbits of the Chinchille strain devided into 2 groups. Identical volumes of 0.5 ml of 10% and 15% sterile medical gelatine solutions were injected in the anterior chamber and 2.0 ml of these in the posterior eye segment of one eye. Cotrol solutions of 0.5 ml 2% methylcellulose and 2.0 ml 0.9% NaCl were injected in the second eye of the animals. The protocol involved clinical, biomicroscopic, ophthalmoscopic, tonometric, histologic and electron microscopic evaluations.
Results. We found that both 10% and 15% medical gelatine solutions, similarly to 0.9% NaCl, did not raise the intraocular pressure, while injected methylcellulose raised it. No toxoallergic and inflammatory changes in the eyes were observed with any of the above solutions. When 10% sterile medical gelatine was applied, the staining solution was eliminated gradually from the anterior chamber for 4.1 ± 0.65 days and from the posterior eye segment for 4.3 ± 0.55 days. For the 15% gelatine solution these elimination times were 5.2 ± 0.45 days and 5.4 ± 0.78 days respectively ($p < 0.001$). The 2% methylcellulose was eliminated from the anerior chamber for 1.6 ± 0.35 days, while the stained 0.9% NaCl solution was eliminated from the posterior eye segment in the course of several hours. We did not find any histologic and electron microscopic changes of the cornea and retina in all eyes tested.
Conclusion. Our study demonstrated that the 15% sterile medical gelatine solution was eliminated slower from the anterior chamber and the posteroir eye segment than the 10% gelatine solution. They did not affect the structure of the cornea and retina They can be used as viscosubstances and as an instrument in the surgery of the anterior chamber and the posterior eye segment (vitrectomy).

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IMMUNOLOCALIZATION OF NITRIC OXIDE SYNTHASE IN OCULAR TISSUES OF FIVE DIFFERENT SPECIES

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Purpose: This investigation was undertaken to examine the distribution of the enzyme Nitric Oxide Synthase (NOS) in normal ocular tissues using specific antibody probes, and to compare the immunolocalization results in multiple species.
Methods: We used both monoclonal and polyclonal antibodies from a commercial source (Transduction Labs) made against peptide fragments to specific isoforms of NOS. Four micron sections of paraformaldehyde fixed, paraffin embedded tissue were processed with the TechMate 1000 robotic immunostainer (BioTek Solutions), using an alkaline phosphatase linked secondary antibody probe.
Results: Staining was highly similar across all species examined (Human, Dog, Cat, Rabbit, Rat) with antibodies specific for the neuronal (bNOS) and endothelial cell (ecNOS) forms of nitric oxide synthase. In the anterior eye structures, staining was observed primarily in the epithelium of the ciliary processes, although immunostaining with the ecNOS antibody was found in corneal and vascular endothelium. In retina, labeling was observed in the ganglion cells, inner nuclear layer and plexiform layers. In addition the ecNOS specific antibody labeled the retinal pigmented epithelium in one species (Dog). No tissue labeling was found when an antibody specific to the induced isoform of NOS was used.
Conclusions: NO is recognized as a multifunctional molecule which can interact with cytokines to exert controlling influences on diverse types of cells. Recent evidence has demonstrated that NO donors injected intracamerally can raise intraocular pressure (Fisher et al IOVS 36:(4)S733). The localization of NOS in the ciliary epithelium suggests a possible cellular mechanism underlying this observation. Immunolocalization of NOS in retinal ganglion cells may indicate that NO has some, as yet unexplained, role in normal retinal physiology. The presence of NO, particularly if upregulated, could possibly explain the dramatic increase in cell death seen after retinal injuries.